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ABSTRACT

A report is given of research conducted to determine the relationship between the response of the human body to stress and the incidence of physical diseases. Seven areas of the nervous system were tested in the study: cardiovascular, electrodermal, gastrointestinal, musculoskeletal, piloerectors, pupilometric, and respiratory. The results of the tests are reported in both narrative and tabular form. It is suggested that this data be used for the evaluation of disease prevention and health promotion activities and programs. (JD)

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The Human Stress Response: An Empirical, Normative Evaluation of Nervous Psychophysiological Paramaters & Dimensions as a Measure of Health

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The accountability and efficacy of health intervention, promotion, or education programs is becoming a critical issue facing health education professionals. This presents a fundamental problem; how do we measure health? To validate professional worth and effectiveness, the central construct of our field must be operationalized.

The term "health" may be defined and quantified along many dimensions. In a biological framework, one critical facet of physical health relates to the individual responses of the human body to stress. Stress reactivity is a powerful factor in physical health and disease. According to Harrison's Principles of Internal Medicine, 50 to 80 percent of all physical disorders are either mediated by or causally related to the experience of stress via psychosomatic interactions. Before stress reactivity may be utilized in operationalizing health, fundamental empirical research must be conducted to determine the nature, dimensions and parameters of the human stress response.

The paper presented here reports the results of an empirical normative evaluation of the nervous psychophysiological response dimensions and parameters of the immediate human stress response.

Purpose

The purpose of this investigation was threefold:

1) To empirically evaluate the human nervous, psychophysiological stress response curve.
2) To determine empirically the critical dimensions of the response.
3) To establish norms across all available, noninvasive psychophysiological response systems in relation to the empirically determined dimensions of the nervous response curve.

Method

The population sample that was used in this investigation was comprised

of 653 subjects, 355 males and 398 females, drawn from undergraduate "Controlling Stress & Tension" classes at the University of Maryland, College Park campus, from the Fall semester of 1977 to the Summer of 1979. Subjects ranged in age from 18 to 57 years, with a mean age of 24. Any subject under regular prescribed medication for hypertension, diabetes, or epilipsy; or anyone using prescribed stimulants, depressants, or phenothiazines; or anyone with previous training in systematic relaxation (i.e. yoga, TM, or biofeedback, etc.) was not accepted for use in this investigation.

All testing was carried out in the Psychophysiological Research Laboratory of the Department of Health Education at the University of Maryland, College Park. All parameters were measured with the aid of a digital Coulbourn Modular Instrument System.

In relation to the selection of measured parameters, seven nervous psychophysiological response systems exist for potential assessment. These include:

* 1) Cardiovascular
* 2) Electrodermal
3) Gastrointestinal
* 4) Musculoskeletal
5) Piloerectors
6) Pupilomaetric
* 7) Respiratory

* indicates systems measured for this investigation.

On the basis of the measurement capabilities of current noninvasive technology, the following four response systems were selected for this investigation and measured, along the parameters indicated:

1) Cardiovascular
    a. Pulse Rate
    b. Pulse Amplitude
    c. Pulse Propagation Time
2) Electrodermal
    a. Skin Potential Response
3) Musculoskeletal
    a. Frontalis EMG
4) Respiratory
    a. Respiration Rate

To transduce the pulse rate and amplitude signals, an infrared photoplethysmograph was located on the palmer surface of the second digit's distal phalanx of the subject's non-dominant hand. Pulse propagation time was assessed by comparing the "P" wave signals from the photoplethysmograph and the ECG "R" wave signals obtained from a V-6 precordial placement, with a sternum ground, of three Beckman silver-silverchloride electrodes. Skin potential response was picked-up using two Bechman silver-silverchloride electrodes placed on the palmer surface of the third and fourth digit's distal phalanx of the non-dominant hand. Frontalis EMG measurements were taken from a bipolar arrangement of three Cyborg silver-silverchloride electrodes, centered 2 cm. over the browline of the forehead, with a 3 cm. intereiectrode distance. Respiration rate was measured vea differential filtration of the precordial ECG signal to pick-up intercostal EMG activity.

The testing protocol typically involved an eight minute stabilization period, followed by a two minute baseline measurement. Following baseline, two stressors were presented individually, with a five minute recovery period following each stimulus. Data was collected anu digitized at six second intervals. The protocol used is illustrated in figure 1.

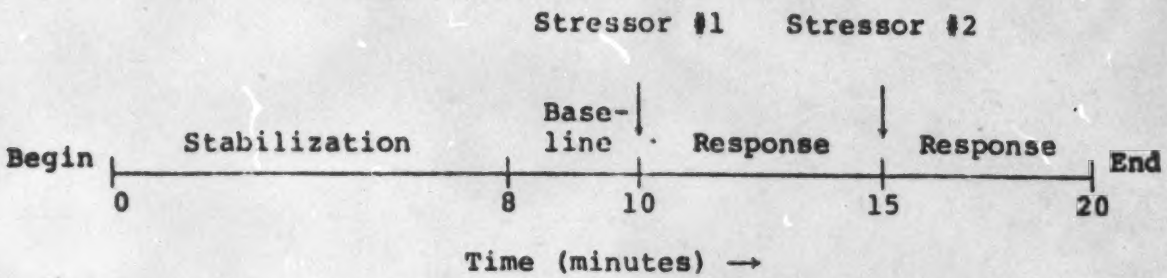

Figure 1. Sequential Outline of Testing Procedure

Two laboratory stressors were utilized to ensure the elicitation of the complete nervous response. The stressors chosen included a rapid onset stimulus (2,700 hz. tone at 90 dB for o.7 seconds), and a cold pressor (dominant hand immersed in 2^{o} centigrade water bath for 30 seconds). These two have been established in previous literature to display relative stimulus-response specificity; one eliciting a cholinergic and the other an adrenergic response, respectively. The stressor's order of presentation was randomly varied and expressed response assessments wer collapsed across the two stimuli for each parameter and response dimension.

Test/retest reliability assessments were made using a one week interval and 36 volunteers, across all parameters and response dimensions used.

Results

The six second data points across all subjects were averaged (arithmetic mean) and graphed for each parameter, in order to reveal the nature of the complete nervous stress response curve. The following set of illustrations display the response curve as it was revealed, and indicate the appropriate response dimensions to include in evaluating nervous stress reactivity.

Previous literature addressing nervous stress reactivity has traditionally focused on two parameters. The first of these is the parameter baseline (figure 2). The baseline value is the resting level of the measured parameter, i.e. heart rate, muscle tension, basal blood pressure, etc.. This dimension represents the systemic activities of the organism at rest, presumable under no psychophysiological arousal. During this investigation, baseline assessments were made for a two minute period following an eight minute rest and stabilization interval.

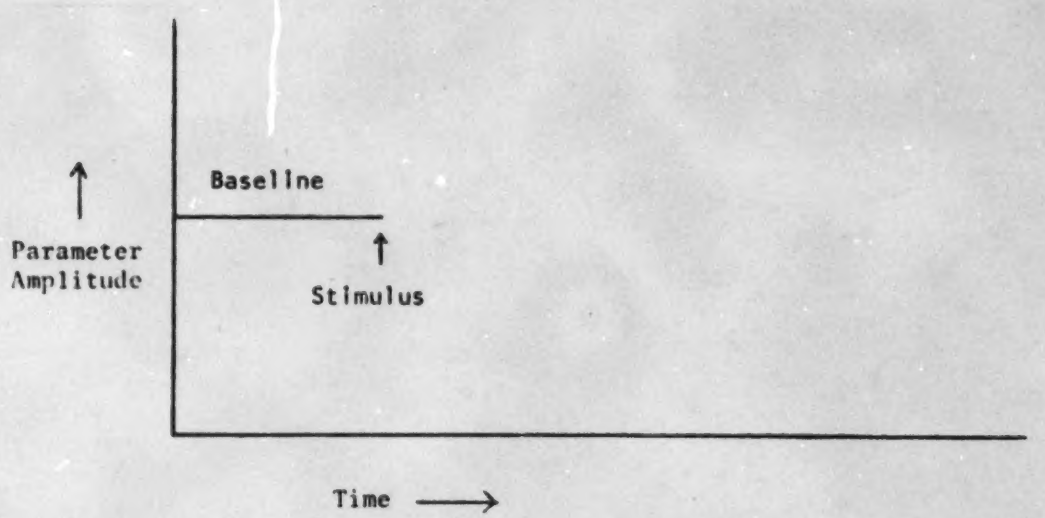

Figure 2: Baseline

Following baseline measurement, a stimulus was presented to elicit an arousal response. Empirical analysis revealed initial manifestations of sympathetic arousal activity across the measured organ systems. This phase of the nervous reaction is labeled the sympathetic arousal response (figure 3).

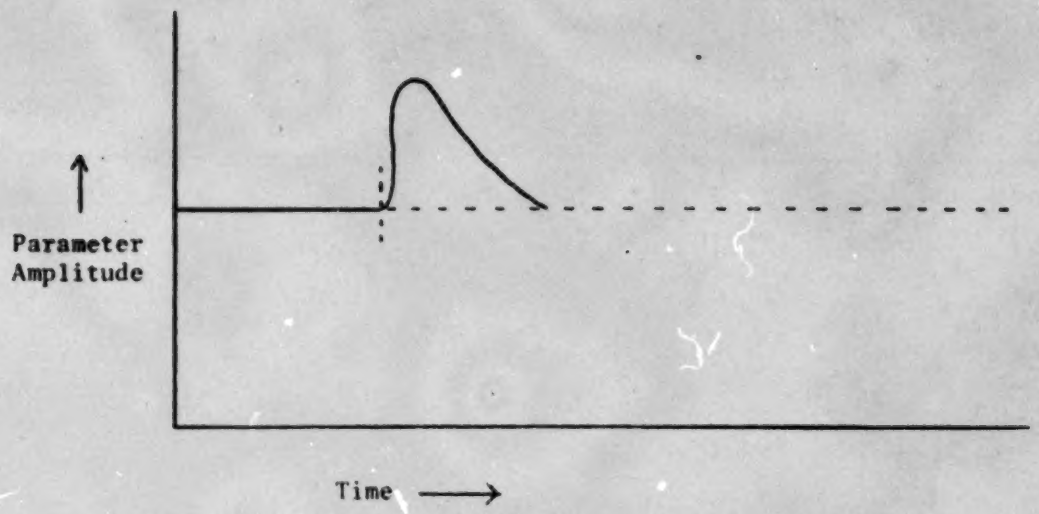

Figure 3: Sympathetic (arousal) Response

It has been the convention in previous stress related studies to assess reactivity on the basis of the amplitude of the sympathetic arousal response. In other words, following the stressor, how high did blood pressure rise, or what was the peak respiratory rate displayed, etc.. Response amplitude therefor represents the second traditionally utilized dimension of stress reactivity, indicating the peak response of an organ system to the stimulus (figure 4).

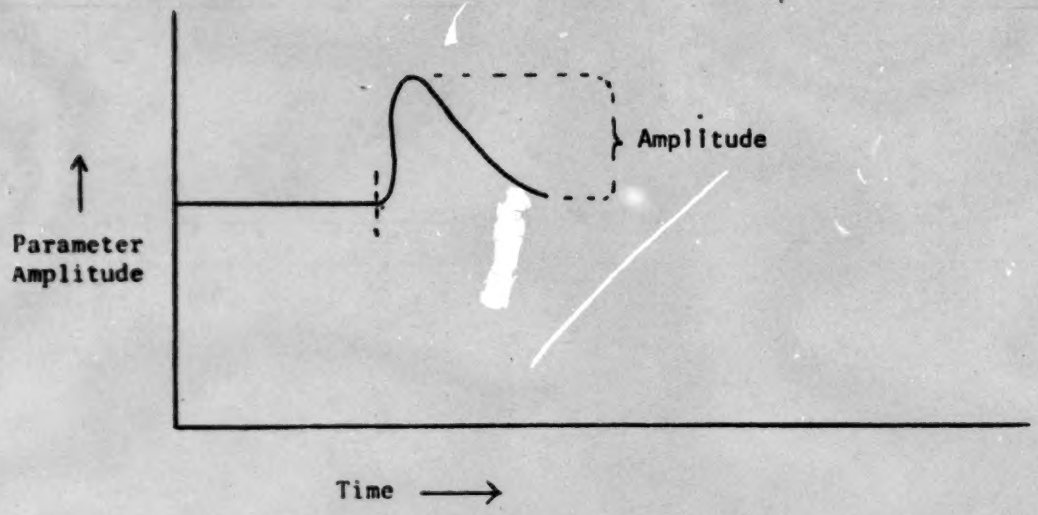

Figure 4: Response Amplitude

If however, specific stress reactivity patterns are to be utilized to predict pathologies, another dimension of this sympathetic arousal response merits investigation. The degenerative processes of most psychosomatic disease etiologies are more linked to the duration of a pathogenic condition rather than the peak amplitude of response to stimuli. Developing a systemic disorder is therefore linked at least as closely to the duration of a pathogenic response pattern as to its peak amplitude. Therefore, a third dimension of the nervous response pattern meriting empirical observation and quantification is response duration. This dimension refers to the time span, from stimulus to recovery, covered by the sympathetic arousal response per measured parameter. In other words, how long was the organ system manifesting higher than baseline arousal following the stimulus (figure 5). Response duration was operationally defined as the time interval from onset of the stressor to the end of the third consecutive measurement of the parameter that is within, or below one standard deviation of the baseline mean.

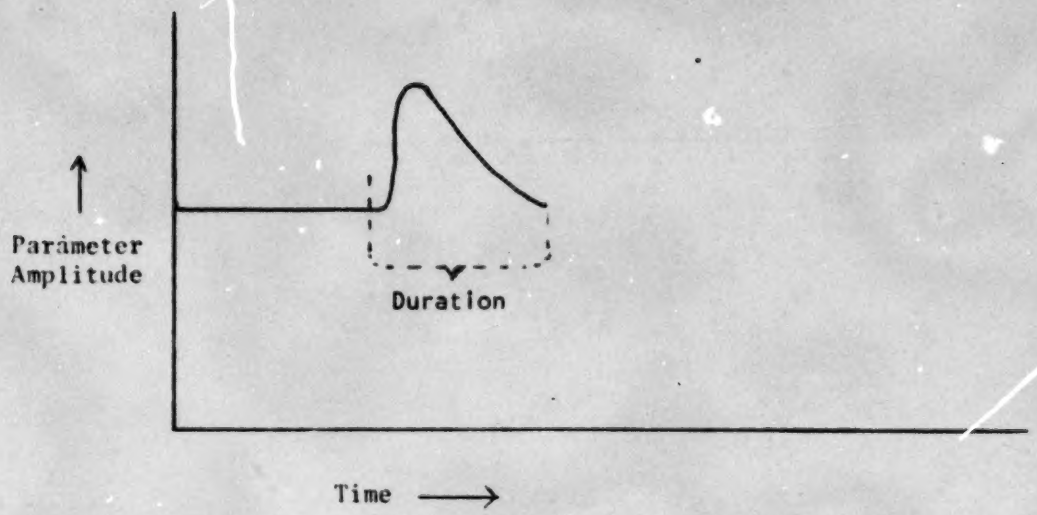

Figure 5: Response Duration

Further empirical observation of parametric recovery following the return to baseline of the sympathetic arousal response, revealed a second phase of the post-stimulus response pattern. After the initial arousal phase, organ activity consistently displayed a significant drop in measured arousal, below the pre-established baseline level. This phase of the nervous response is termed the parasympathetic rebound (figure 6).

The nature and significance of this "rebound" phase is of great interest. It appears to represent an effort on the part of the organ system, or parameter, displaying the response to offset the previous sympathetic arousal activity. It appears to be a type of nervous mediated homeostatic mechanism. As described early in this century by Cannon, this process represents the internal activities of the organism to maintain balanced functioning of organ systems around a tolerable mean activity level. The parasympathetic rebound may then be the organ systems' attempt to restore global, dynamic, functioning balance to the effected organ systems.

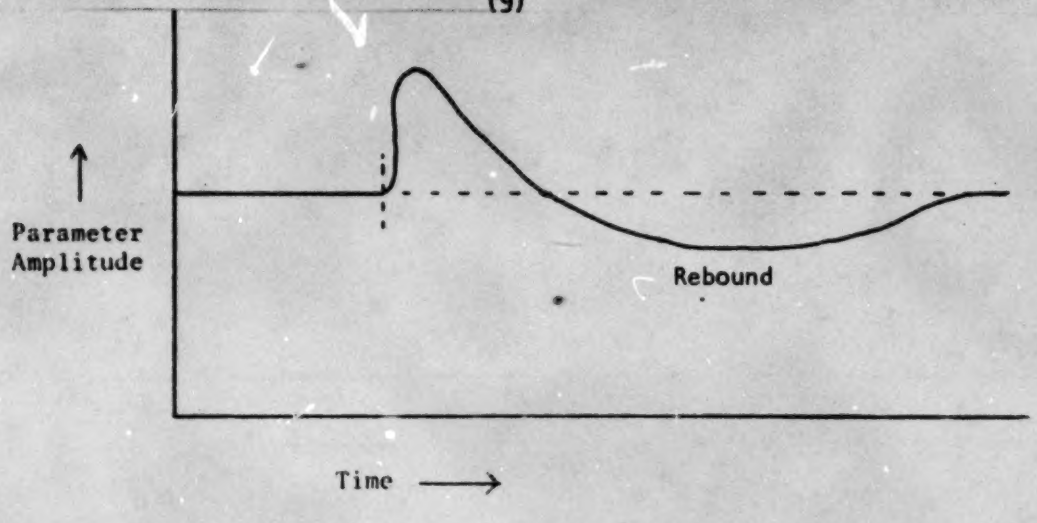

Figure 6: Parasympathetic Rebound

If we assume that a systemic disease condition will result from an imbalanced state of an organ system, that is the system displaying excessive activity in a hyper or hypo aroused state, then this rebound assessment may be of ultimate significance im predicting pathologies. A healthy condition would be one in which an optimal balance between the two antithetical innervation systems is being displayed by the organ's complete response pattern.

This systemic balance may be operationalized by evaluating the magnitude of sympathetic activity compared to the magnitude of the resultant rebound activity. This relationship may then be expressed as a retio score of measured sympathetic to parasympathetic activity. Diagramatically, quantification of this response dimension may be illustrated as follows (figure 7):

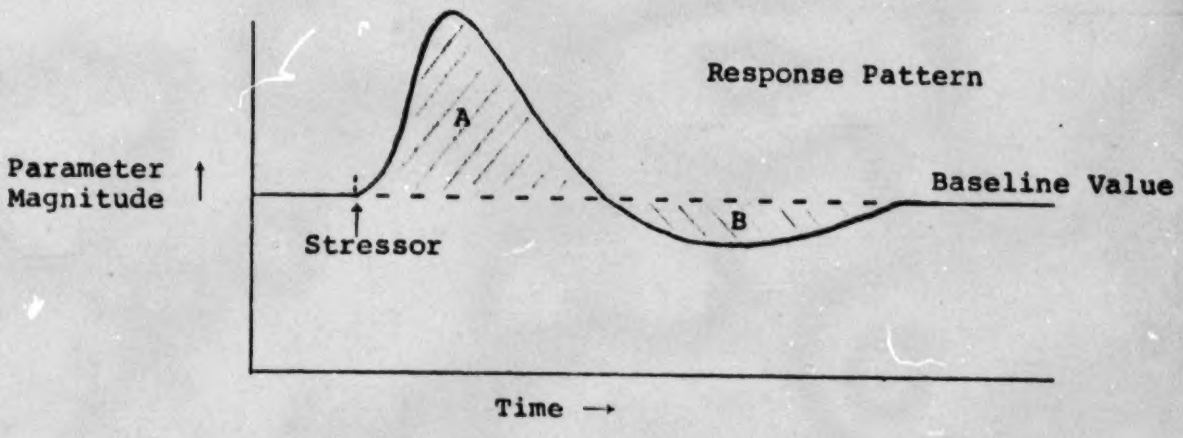

Figure 7. Sympathetic/Parasympathetic Ratio Assessment.

Since most of the measured parameters were not monitored continuously but in six second intervals, calculation of the S/P response ratio was done with the aid of the following formula:

$$\frac{\sum_{i=0}^{B1} (X_s - \bar{X}_B)}{\sum_{i=B1}^{B2} (\bar{X}_B - X_p)} = \text{S/P Response Ratio}$$

where: $\bar{X}_B$ = Baseline Mean

X_s = Arousal Response Values

X_p = Rebound Values

O = Stressor Onset

B1 = Moment of First Return to Baseline

B2 = Moment of Second Return to Baseline (maintained for three consecutive measurements within one standard deviation of $\bar{X}_B$)

Therefore, empirical analysis of nervous stress response curves has revealed the existence of the following four response dimensions:

1) Baseline
2) Response Amplitude
3) Response Duration
4) Sympathetic/Parasympathetic Response Ratio

Normative assessments were made on each response parameter (frontalis EMG; pulse rate, amplitude, and propagation time; respiratory rate; and skin potential response) across all four empirically derived response dimensions. The normative findings are summarized in table 1.

Table 1. Normative Data: Means and Standard Deviations for Parameters and Response Dimensions During Initial Testing

	Response Dimension			
Parameter	Baseline (see below)	Amplitude (see below)	Duration (# of Print Intervals)	S/P Ratio
EMG	$\bar{X}$ = 3.7	4.3	4 (24 sec)	3.6
	S_X = 1.2	1.7	2 (12)	2.4
PR	2307	2022	7 (42)	2.7
	108	307	4 (24)	1.2
PA	565	547	11 (66)	2.1
	216	309	6 (36)	1.7
PPT	987	721	9 (54)	3.9
	138	102	7 (42)	2.6
SPR	2	4	2 (12)	4.9
	1	3	1 (6)	1.1
RR	13.2	18.9	6 (36)	2.8
	2.6	4.3	2 (12)	1.8

For Baseline and Amplitude dimensions, the following units of measurement apply to each parameter:

EMG: microvolts (average RMS)
PR: milliseconds (3 P-P intervals)
PA: millivots (3 pulse curves)
PPT: milliseconds (3 R-P intervals)
SPR: skin potentials (in 6 seconds)
RR: cycles per minute

The initial testing measurements were compared to measurements obtained one week later on all subjects for each parameter and response dimension. Table 2 presents the Pearson product-moment correlation coefficients for each dimension of each parameter, initial test to one week retest, as a way of assessing the test/retest reliability of the protocol.

The test/retest correlations revealed that three of the cells failed to display significant correlations. These included pulse amplitude baseline, skin potential response duration, and respiration rate amplitude. All other test/retest correlations were significant at the $p \leq .05$ level. Overall, the highest test/retest reliability values were obtained for the response duration dimension and the frontalis EMG parameter. The S/P ratio was the only dimension to display significant correlations across all parameters, although the correlations for SPR and RR were rather low, +0.35 and +0.38, respectively. The response amplitude displayed the lowest correlations of the response dimensions. All of the correlations were below 0.60 and five of the six parameters were below 0.50 for this dimension. Three parameters had significant test/retest correlations across all response dimensions. These included frontalis EMG, pulse rate, and pulse propagation time.

Table 2. Test/Retest Reliability: Pearson Product-Moment Correlation Coefficients for Parameters and Response Dimensions, Initial Testing to Second Testing Exposure

	Response Dimension			
Parameter	Baseline	Amplitude	Duration	S/P Ratio
EMG	+0.87*	+0.58*	+0.67*	+0.64*
PR	+0.63*	+0.44*	+0.59*	+0.55*
PA	+0.14	+0.36*	+0.56*	+0.79*
PPT	+0.61*	+0.47*	+0.74*	+0.64*
SPR	+0.82*	+0.35*	+0.32	+0.35*
RR	+0.52*	+0.28	+0.75*	+0.38*

Redundancy in dependent variables was analyzed by computing Pearson product-moment correlation coefficient matrices between parameters collapsed across response dimensions and then between response dimensions collapsed across parameters for standardized initial testing measurements. Table 3 summarizes the intercorrelations between parameters and Table 4 summarizes the intercorrelations between response dimensions.

Table 3. Redundancy in Dependent Variables: Pearson Product-Moment Correlation Coefficient Matrix Between Response Parameters

	Parameters					
	EMG	PR	PA	PPT	SPR	RR
EMG	+1.00	-	-	-	-	-
PR	-0.34	+1.00	-	-	-	-
PA	-0.19	+0.43	+1.00	-	-	-
PPT	-0.28	+0.36	+0.48	+1.00	-	-
SPR	+0.11	-0.15	-0.32	-0.07	+1.00	-
RR	+0.23	-0.39	-0.19	-0.43	+0.12	+1.00

Correlation coefficients were computed on standard scores (t-scores) for initial testing values.

Table 4. Redundancy in Dependent Variables: Pearson Product-Moment Correlation Coefficient Matrix Between Response Dimensions

	Response Dimension			
	Baseline	Amplitude	Duration	S/P Ratio
Baseline	+1.00	-	-	-
Amplitude	+0.17	+1.00	-	-
Duration	-0.23	+0.32	+1.00	-
S/P Ratio	+0.13	+0.29	+0.47	+1.00

The criterion for determining redundancy in dependent variables was a correlation coefficient in excess of 0.80. Intercorrelations across all parameters used in this investigation revealed that no two parameters were correlated higher than 0.50, a value far below the 0.80 redundancy criterion. The same results were the case for the response dimensions. This indicates that each of the response parameters and dimensions measured in this study made a unique, independently significant contribution to the overall assessment of human, nervous stress reactivity.

Future Research

Three logical extensions of this work may be suggested for future investigation which would take the empirical findings presented here and utilize them in applied settings.

1) Utilizing the normative data it may be possible to develope a standardized stress assessment protocol to be used in predicting the future onset of stress related disorders, by evaluating an individual's pattern of stress reactivity and identifying over reactive or weak organ systems.

2) The normative data may be correlated with disease incidence to ultimately facilitate a quantifiable risk assessment tool for evaluating stress related disease risk for a given individual.

3) The findings of this envestigation may be utilized to establish a theoretical base and operational assessment criteria for evaluating the efficacy of stress management and intervention techniques, or programs.

Prior to future applied research, the next logical investigative step is epidemiological. This would involve gathering disease incidence data on a population also measured on the psychophysiological protocol established here, to determine which components of measured stress reactivity may serve to help predict disease.

Conclusion

The existence of this normative data opens the way to more articulate applications of nervous stress reactivity for the assessment of psychosomatic disease potential, health, and efficacy evaluations of disease prevention and health promotion activities and programs.

Bases on this fundamental empirical research, it may now be within our grasp in the near future to develop and validate an objective procedure for predicting future pathologies, well in advance of their actual occurance, assessing somatic risk in individuals due to early physiological response predispositions to stress, and evaluating the psychophysiological efficacy of diverse stress management or health intervention techniques.